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ORIGINAL ARTICLES

The Broad Symptomatologic Spectrum In Injuries of the Cervical Spine

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It has long been known that injuries of the neck can produce manifestations in other parts of the body. The paralysis of the legs that appeared when the neck was broken was easily associated with injury of the cervical spine.

On the other hand, other groups of symptoms, less dramatic in their onset and frequently delayed in their appearance, were not so readily attributed to injuries of the neck until relatively recent times. In the past few decades, partly as a result of the "whiplash" injuries of automobile accidents, it has been recognized that a wide variety of symptoms can be associated with these injuries.

A great deal of study has been directed to the pathologic processes and to the neurologic involvement. As time passes and enlightened experience is gained, newer manifestations are found that must be attributed to these injuries. Some of the clinical pictures are so unexpected that it may be worth reviewing the subject.

Thumbnail Sketches

To illustrate, let us begin with a few thumbnail sketches of case histories.

1. A man in his mid-forties came in with the complaint, "I must have broken a rib". He reported that while drying his back after his morning shower, he suddenly experienced a sharp stab of pain in his right chest.

Some pain persisted, but this was sharply exaggerated when he moved the upper part of his body. A careful examination was made of his thoracic cage, but no fracture could be found in his ribs.

His neck was then examined and here one found muscle spasm, limitation of motion, and marked tenderness on pressure over the right articular processes of the fifth and sixth vertebrae.

An x-ray of the cervical spine showed considerable arthritic spurring. The neck was immobilized and in a few days the pain in the chest cleared up.

Chief Complaint

2. Another man in his early forties had as his chief complaint a disturbance in the appearance of his left eye. His history revealed that for a number of months preceeding his visit, he had had some discomfort over his heart and that he had gone to a physician for an examination.

The physician told him that his heart was in good condition and that the symptoms were those of a myositis, so that he might do well to consult an orthopedist. Because his pains were not severe he did nothing about this until one day he notnced that his left eye-lid drooped. This drove him into seeking a consultation.

Examination revealed a typical Horner syndrome,-miosis, enophthalmos, and drooping of the eye-lid. His head was carried forward and all of the movements of the lower cervical spine were markedly restricted.

X-rays of his lower cervical vertebrae showed marked arthritic spiculation and some of the spurs on the bodies protruded posteriorly.

Physical therapy for the neck was instituted and in a short time the precordial pains cleared up. The patient left the city at about that time and the follow-up of the Horner syndrome was not obtained.

Feared Cancer

3. A young woman came in complaining that

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she was afraid that she had cancer of the right breast. These fears were based on the fact that there was pain in the breast itself.

On examination it was found that she was overweight and had rather pendulous breasts, but no abnormal mass could be felt, nor was there any unusual local tendernes in her breast tissue.

She carried herself so that her head jutted forward, the trapezius muscles were tight, and bending to the left and hyperextension were quite restricted. Pressure over the sixth cervical vertebra on the right articular process gave severe pain.

Treatment was directed to the cervical spine and in a very few days the pain in the breast cleared up.

Right Arm Paralysis

4. A seventy-four-year-old man was sent in for examination and treatment of a paralysis of the right arm. His story was that eighteen days earlier, he was riding in an automobile that was struck in the back.

He was jolted, but he had no particular pain at that time. That evening, however, he developed a stiffness in the neck and a little bit of pain.

There appeared a weakness in the right arm and soon he was unable to raise his arm. Examination of his arm revealed a rather marked weakness of his deltoid, biceps, and supinator muscles.

No sensory loss was made out. In his neck there was marked spasm, restriction of motion and tenderness over the sixth articular process on the right side. Pressure at this point gave pain that was referred to the right shoulder and upper arm.

X-rays showed considerable narrowing of the interspaces between C5 and C6 and between C6 and C7. Treatment consisted of protection of the neck and the use of physical therapy for this region. Within two weeks, most of his power had returned.

Twist of Neck

5. A fifty-eight-year-old man, following a mild twist of his neck, developed a pain in his neck and a weakness in his left arm. Several months later his left leg became weak.

He showed an atrophy of the thenar and hypothenar eminences of his left hand and a weakness of the left leg that led to a mild hemiplegic gait. The reflexes were normal. His cervical spine presented marked osteoarthritic changes.

He was followed for several years during which time his leg weakness increased. On the assumption that he had a spondylosis, he had a laminectomy of the cervical spine and a release of the dentate ligaments.

Following this, his leg weakness eased substantially.

Symptoms Dissimilar

In these five patients, not only were the symptoms dissimilar, but the areas complained of were in different parts of the body. The common denominator was pathology of the cervical spine.

Is there any satisfactory explanation for so many syndromes originating in one area? Yes. We should be able to understand the mechanisms if we look into the anatomy of the cervical spine and its contiguous structures.

This area is analogous to a telephone exchange room in which there has been an explosion. The room itself would show damage.

On the other hand, a number of disturbances would appear in various parts of the building controlled by the switchboard. The silencing of telephones, the ringing of alarm bells, and other complaints would be reported from near and far.

By collecting these complaints and correlating them with the plan of the wiring, the repair man would be able to identfy the location of the trouble spots.

Complex Unit

The cervical spine, like this telephone exchange room, is a complex unit harboring many nerves that control areas near and far in the human body.

Any severe damage in the neck may bring about pain in the neck, but may also cause disturbances in the various parts controlled by these nerves.

To better understand the development of these disturbances, a review of the anatomy of the cervical spine is in order.

ANATOMY OF THE CERVICAL SPINE

In essence, the cervical spine is a firm, though flexible, structure surrounding and protecting the spinal cord. The protective quality is inherent in its bony rigidity. The flexibility is produced by its being divided into segments which are separated from each other by elastic inserts. The whole structure affords a fairly strong though flexible armor to protect the spinal cord.

The spinal nerves that run from the spinal cord through this flexible armor to extra-spinal areas are also given fairly solid protection. The nerves emerge through channels in the elastic interspaces, but are protected from compression forces

by bony projections that reach downward and upward from each vertebra.

These projections articulate, and their junctions are maintained by join structures with synovial membranes and firm ligamentous capsules. The nerves lie directly in front of these joints so that they are given bony protection posteriorly as well as superiorly and inferiorly. Anteriorly, the channels are completed by intervertebral disks and parts of the vertebral bodies.

Intervertebral Foramina

These channels, the intervertebral foramina, are from ¼ to 1/6 of an inch in diameter, whereas the spinal nerves are from 1/12 to 1/6 of an inch in diameter. The free spaces are filled by loose areolar tissue that also contains vessels.

We thus have a remarkable structure, one that allows considerable motion and yet serves as a solid protective armor for the vulnerable nerve tissues under normal conditions.

Under abnormal conditions, however, this armor may not only fail to protect the nerve tissue but may actually serve as a medium of damaging it. Under a force of sufficient magnitude to break the bone or to disrupt the articulating areas, the rigid casement becomes a shearing or compressing medium that damages the enclosed nerve tissue.

Damage By Vertebrae

In a fracture or dislocation of the spine with displacement, the vertebrae can cut the spinal cord, or in lesser displacements can compress the nerves in the intervertebral foramina.

At the points of movement severe strains may cause soft tissue damage which, though not visible on the x-ray, produce sufficient local swelling to interfere with the function of the nervous tissue.

Actually, even minor injuries, if repeated often enough, and over a long period of time, can produce degenerative changes which through the formation of cartilaginous projections and bony spurs will reduce appreciably the clearance needed for the spinal nerve.

INJURIES

In practice, we find a number of injuries that may cause damage to the nerve structures.

A. Affecting the spinal cord.
   1. Fracture of the cervical spine with major displacement.
   2. Dislocations of the cervical spine with major displacement.
   3. Dislocation (with cord injury) spontaneously reduced.

B. Affecting the intervertebral nerves.
   1. Fracture of the neural arch of the vertebra.
   2. Dislocations of subluxations, unilateral or bilateral.
   3. Disk compression
      (a) With immediate herniation
      (b) With tearing that leads to delayed herniation.
   4. Sprains of the intervertebral joints, as in whiplash injuries or paratrooper's neck, with swelling of capsules.
   5. Cumulative strains, leading to osteoarthritis, etc.

C. Avulsion of the nerves.

These injuries may give pain that is localized in the neck. In addition, they may produce remote effects by affecting the nerve tissues in the following manner.

1. Spinal cord.
   a. Section of the cord, complete or partial.
   b. Cord compression.
   c. Concussion.
   d. Localized irritation by spurs, projecting into the canal.
2. Spinal nerves.
   a. Compression in the foramina by fractures or dislocations.
   b. Irritations by swollen capsules, arthritic spurs or herniated disk tissue.
3. Sympathetic nerve fibre damage.
4. Blood vessel irritation.

PATHOMECHANICS OF PERIPHERAL PHENOMENA

The effects of major cord damage with peripheral paralysis, spastic and flaccid, sensory disturbances and loss of bowel and bladder control are so well known that they need only be mentioned here.

The localized irritation of the spinal cord by spurs projecting into the spinal canal has received attention only recently and has been reported under the name "Spondylosis".

Under this title there have been described clinical pictures with bizarre arrays of symptoms affecting the lower extremities as well as the upper limbs.

Changes suggestive of multiple sclerosis and even amyotrophic lateral sclerosis have been described. The signs and symptoms have not as yet been formulated into a crystallized syndrome.

It is quite likely that a large group of authentic cases will have to be reported before a definite pattern is evolved.

Disturbances

Pressure on the nerve roots has been known to produce **sensory** disturbances when **sensory** fibres are irritated, and **motor** disturbances when **motor** fibres are affected. This explanation, however, is not adequate for all observations.

For example, it was found that some patients had precordial pain, like angina pectoris, that could be produced by nerve irritation in the neck.

It would be simple to explain this by saying that the stimuli were affecting the anterior medial thoracic nerve, innervating the pectoralis major, and the anterior lateral thoracic nerve, innervating the pectoralis minor.

But these nerves are classified as motor nerves! Study revealed that protopathic pain, a discomfort that can be quite severe, but is not sharply demarcated, can be projected along compressed motor nerves.

With this explanation one can account for the pseudo-angina pectoris that develops in a patient following injuries of the cervical spine.

Nerve Compression

The mechanism for the production of the stimuli we have been discussing has been nerve compression in the intervertebral foramen. There is, in addition, evidence that referred pains can be produced by irritative lesions of the intervertebral joints and disks without actual compression of the nerve roots.

We know, for example, that children with hip involvement, such as a slipping of the capital epiphysis of the femur, will complain of pain in the knee along the course of the obturator nerve, even though this nerve does not run through the joint where it could be compressed by the displacement.

We believe that this phenomenon is produced as follows. The capsule of the hip joint is innervated by fibres from the obturator nerve.

When these fibres are irritated, the message is sent to the brain where it is interpreted as coming from the obturator nerve ends, so that it is attributed to the knee.

Plexus Dissected

In the cervical spine it has been possible to dissect the plexus to its roots under local anesthesia.

In instances where the intervertebral foramina were exposed and one could determine that there was no compression on the roots, one could produce pains referred down the arm by sticking a needle into the intervertebral joint or into the disk.

The sympathetic nervous system is affected in two ways. Pressure on the roots in the intervertebral foramina also catches the rami communicantes which run through the roots and separate shortly after they leave the foramen to connect up with the sympathetic nervous chain.

It is injury to these roots that produces the Horner syndrome. It is also said that the sympathetic nervous supply to the vascular tissues can be irritated by abnormal pressure and so give peripheral manifestations. Vasospasm and the scalenus-anticus syndrome are said to originate in this way.

CLINICAL MANIFESTATIONS

Let us now consider the various clinical pictures that are met. Most of the syndromes that will be presented have actually been observed by your speaker. A few of those that are listed have not been entirely convincing as to the association between the injury of the cervical spine and the peripheral manifestations, but they have been reported by capable observers so that they are listed here.

1. The most serious clinical pictures are, of course, those that involve crushing of the cord in the cervical area. Here we are faced with early death, or if the patient recovers from the immediate injury, with paralyses involving the extremities and frequently with a loss of bowel and bladder control.
2. **Occipital headaches.** It is redundant to say that such headaches can occur as a result of many factors, and patients should be studied from many angles. Since pressure on the first cervical nerve can cause radiation into the occipital region, orthopedic consultation should be included. In a significant number of such patients, these headaches have been found subject to control by the therapeutic test of head traction.
3. **Temporal pain.** It is not uncommon to have patients come in with a complaint of pain that runs from behind the ear, over it into the temple and even as far as the orbital area. Such pains follow the course of the auricular nerves which originate chiefly in the second and third cervical interspaces.
4. **Drooping eyelids.** When this symptom is associated with a small pupil and enophthalmos, one has the Horner syndrome. This can be produced by irritation of the sympathetic nervous system which receives rami communicantes from the lower cervical nerves.

5. **Neck pains.** Such discomforts are easily associated with pathology of the cervical spine. It must be emphasized that these pains are often associated with peripheral manifestaions but the absence of pain in the neck does not rule out pathology in the neck. As an anology one may remind you that frequently aciatic pains in the leg, that originate from nerve irritation in the lower spine, may be present without actual complaint of low back pain.

6. **Shoulder pains.** It is surprising how often patients come in with complaints of pain in the shoulder and yet show no evidence of shoulder pathology. Frequently they report that they have had physical therapy for this joint or even injections of Hydrocortone and x-ray treatments without relief. An examination of the neck will often show the area of involvement to be the cervical spine.

7. **Pain in the arm.** Pains in the arm that originate in the cervical spine follow a course that is not limited to one of the three major nerves. They run down the arm frequently much lower than the elbow as contrasted to the pains referred from a subdeltoid bursitis.

8. **Paralyses of the muscles of the arms and fingers.**

9. **Numbness and tingling in the wrists and fingers.** When such symptoms are found, the cervical spine must be suspect, particularly when the distribution of the sensory disturbance does not follow the exact pattern of one peripheral nerve.

10. **Angina-like pains.** Pains in front of the heart naturally lead one to a careful examination of the heart. It is, however, fairly well established that such symptoms can originate in the cervical spine.

11. **Pleuritic-like pains** are often presented and yet no evidence of lung or pleural involvement can be found. One may find the explanation for these pains in the cervical spine.

12. **Fractured rib pains.** It is surprising how frequently patients who have been in an automobile accident come in with a complaint of pains in the ribs and yet a careful examination of the ribs clinically and by x-ray is negative. Such pains can be felt when the roots of the nerves between the lower cervical vertebrae are compressed.

13. **Pains in the breasts.** A case report mentioned earlier in this paper indicates the association.

14. **Pains over the kidneys.** It is also surprising how low the symptoms of cervical root compression can be projected, particularly along the longissimus dorsi nerve.

15. **Weakness in the lower limbs.** Although many more case reports are necessary to crystallize the clinical picture of lower extremity discomforts originating from irritation of the spinal cord by spurs projecting into the canal, sufficient evidence has already been presented to make it necessary for us to consider this relationship.

CONCLUSIONS

The cervical spine, when disturbed, can be responsible for a number of apparently unrelated clinical pictures. A number of such manifestations has been presented and the anatomic and physiologic links have been described.

Many of these symptoms can be produced by local pathology, but if a careful examination fails to reveal objective local signs to account for them, the physician will often find it rewarding to examine the cervical spine.

University of Colorado Schedules Conference on Edema

A three-day post-graduate conference on "Edema—Its Pathogenesis and Management" has been scheduled March 13-15 at the University of Colorado Medical Center, Denver.

The conference will be devoted to basic considerations and clinical applications of kidney function, edema and diuresis. It is designed to present in a comprehensive manner the problems of pathogenesis and management of edema as variously encountered in clinical medicine. Special emphasis will be placed on treatment.

Five distinguished guest lecturers and members of the University of Colorado School of Medicine faculty will participate.

A detailed program and further information may be obtained by writing: The Office of Postgraduate Medical Education, University of Colorado Medical Center, 4200 East Ninth Avenue, Denver 20, Colorado.

Emotional Factors in Rheumatic Diseases: Their Role and Treatment

By Peter J. Warter, M.D., *Chief of Department of Medicine, William McKinley Memorial Hospital, Trenton, New Jersey; Assistant Professor of Medicine, Hahnemann Medical College, Philadelphia, Pennsylvania*

This is the seventh and last of an outstanding series of articles covering the effects of emotional stress in the practice of medicine. The articles were prepared for a symposium delivered in January at the Tufts-New England Medical Center in Boston.

SOUTHWESTERN MEDICINE wishes to take this opportunity to express its great appreciation to the Bulletin of the Tufts-New England Medical Center and its editor, Dr. Robert P. McCombs, for making this important series available for publication.

Rheumatic disorders are of such frequent occurrence that they may be classified as the most common of chronic diseases. Relatively few individuals go through life without having a rheumatic syndrome at some time or another.

Arthritis rarely influences the life span of a patient; however, the disability it produces often makes the patient a lonely individual and affects his social and economic circumstances adversely. Constant misery and curtailed activities tend to tax his "good nature" and make him bitter.

Important Factors

Emotional and psychic disturbances are recognized as important factors in rheumatic diseases. Some observers[1] believe it is the most common aggravation of the rheumatic syndrome.

It has been observed that many patients undergoing emotional stress often exhibit a muscle hypertonia which may cause, and almost always aggravates, musculoskeletal symptoms.

It is frequently noted that these symptoms are alleviated when mental tension is lessened. It would seem rational then that treatment that exerts a beneficial effect on mental stress should prove of value in relieving some of the physical symptoms and in increasing the patient's ability better to understand and tolerate his illness.

The susceptibility of soft tissue to distress associated with the rheumatic problem may be enhanced by physical or emotional trauma, environmental factors, dietary deficiencies, infection and fatigue.

Muscle Tension

Emotional trauma appears to produce local neuromuscular or vascular effects with muscle tension that seem to come and go with the rise and fall of emotional states.

By whatever criteria the behavior pattern of emotionally disturbed patients is measured, the same general conclusion is arrived at, namely, that many individuals have apparently been conditioned from early life to live with taut and tense musculoskeletal systems and with capillary systems under intermittent constriction.

These phenomena are affected by emotional states that hyperactive the hypothalamic vegetative system and the adrenals.

Fear accelerates the metabolic and especially the catabolic processes of cells. Worry also dissipates energy. The deprivation of biologic essentials, accompanied by stress situations beyond the threshold of tolerance, definitely aggravates the disease state.

Several Fears

Every arthritic patient experiences several fears: fear of crippling, economic instability, social deprivation, and disinterest on the part of the family. When an individual is repeatedly subjected to environmental patterns unsuited to his temperament, and concerning which he develops an anxiety tension state, his fear automatically causes him to go on guard. Fear produces muscle tension. This leads to what has been termed neuromuscular hypertension, which appears to be related to the fatigue state.

Fatigue is probably the greatest factor in the genesis of disease. Fatigue may be manifested by such sensations as weariness, lassitude and a decreased capacity for mental or physical tasks. These phenomena are a reflection of the body's functional inefficiency and an indication of its susceptibility to injury.

Fatigue produces weakness that reduces the threshold at which effort produces fatigue. Tension does not allow muscles to relax. Under ten-

sion which produces spasm, every motion is a force against a residual spasm, which eventually results in varying degrees of tissue injury.

Individual Museum

The individual is a museum of delicately balanced physiological reactions. Any break in the physiological balance may lead to permanent tissue damage and even invalidism. The symptoms manifested are an expression of the body's attempt to re-establish balance.

The integrity of the capillary system has received very little consideration. It has been shown that soft-tissue changes associated with rheumatoid arthritis affect the normal constitution of the capillary system. Emotional disturbances have been shown to be associated with capillary changes.

There are occasional arthritics whose disease is in remission, but who show visual evidence of the disease. As a defense mechanism against undesirable situations or tasks, they produce false symptoms and restricted motions with which they are all too familiar. They are often able to fool the most astute observer. These problems are occasionally seen in malingerers in compensation cases and in families where sympathy and attention would be otherwise lacking.

TREATMENT

In the management of the patient with rheumatic disease, the physician must be careful not to assign undue importance to one etiologic factor, even though it appears to have an undisputed connection with a particular symptom complex, and to call its environmental biologic relation absolute. A multiplicity of factors is involved in rheumatic diseases.

We are called upon to alleviate pain and to relieve the anxiety state. These patients are fearful, apprehensive and at times very unstable. It is essential to obtain a detailed history of their background — their family, associates, religion, economic status, nature of work and so on. The patient must be viewed and treated as a total entity, and not as a single symptom. His problems should be of as much importance to the physician as they are a concern to the patient. A tense and anxious patient is not in a favorable situation thoroughly to understand the explanations and instructions given him.

Pain Threshold

The pain threshold varies with each individual. The greater the emotional disturbance the lower the threshold. These individuals use dynamic words to try and convince one of the severity of their distress.

The response of the patient to a tried and true therapeutic program will be more profitable if he can be conditioned to positive thinking. The patient's desire to get well is the crux of a successful therapeutic program.

There have been innumerable approaches to aid in treating the emotional factors of the rheumatic problem. Treatment by psychotherapy must be divided into two groups.

In one, the patient is morose with an impending depressive psychosis; in the second, the emotional states are readily discernible and their conflicts not too deep.

Most all rheumatics are in the second group. The first group of patients must be recognized early and treatment by a psychiatrist instituted.

Patients in the second group must first be made acquainted with their problem by a realistic and reassuring approach. Such a patient must be impressed with the fact that no therapeutic program will be of lasting value unless he allows it to be, and that no drug will work beneficially unless he allows it to do so. He must accept every phase of the treatment as an essential and integral part of his desired recovery.

"Dramatic" Cures

Many of the so-called "dramatic" cures are the result of a conquered frustration or inhibition. The improvement continues if the psychic overlay does not return. When the patient and the physician are fortified with this knowledge, it is safe to embark upon other phases of treatment.

Hypnotherapy has been of definite aid in determining the degree of function possible in questionable cases. It has aided a few arthritics to overcome some of the inhibitions and fears which have plagued them.

I use the word "few" justifiably because this procedure should be used only when the patient has been thoroughly appraised by a psychiatrist. The treatment by hypnosis should be given only by a psychatrist trained in this art.

Chemotherapy may be dramatically effective, but few drugs are equally effective in all patients, even if the patients suffer from the same type of rheumatic disease.

Improved Mood

d-Amphetamine sulphate administered once daily by means of a sustained release capsule*[2] has been shown to be an adjunct to other forms of therapy. In many patients, the improved mood produced by this medication has alleviated depression and anxiety.

An interesting and surprising fact was the beneficial effect achieved by nighttime administration

*Dexedrine Spansule® (Smith, Kline and French)
†Hesper-C® (National Drug)

of the sustained release capsule. Some patients whose symptoms were more pronounced in the morning found that taking the capsule shortly before retiring did not prevent them from sleeping and resulted in greatly lessened morning pain and stiffness.

The disturbance of the capillary integrity referred to has been aided remarkably by the use of a capsule† containing hesperidin, 100 mg, and ascorbic acid, 100 mg, in doses of 1 to 2 capsules three times daily. Experience with this combination over a period of years has supported our concept that the correction of capillary defects enhances the total therapeutic regimen in arthritis.

Sedation Employed

Sedation has been commonly employed in attempting to bring about emotional stability in rheumatics. It is generally agreed that sedatives in various forms must be cautiously used, because people habituate to them more easily than is realized. Sedatives do not, as a rule, improve the reasoning of the patient, but they may temporarily raise the threshold of tolerance to his disease, so that he may better cope with its problems.

The past few years have seen the development of "tranquilizing" or ataractic drugs. D a n g e r lies in their misuse and abuse. The most common danger, according to Dickel and Dixon,[4] is that some essentially normal people (1 in 5) taking tranquilizing agents have been "precipitated into psychiatric illness by misinterpretations of their reactions to tranquilizing drugs."

I believe the value of these compounds does not lie in their so-called "tranquillizing" effect alone. The thinking and reasoning of a patient may be more easily molded and conditioned while taking these agents. It is during this period that the physician may be able to instill more confidence and establish a more cooperative desire to carry out his instructions.

Combination

I have reported on the use of a combination of 2,2 diisopropyl-4 methanol-1,3, dioxolane,‡ 25 mg, and reserpine, 0.25 mg, with definite evidence that this therapy results in better understanding and cooperation on the part of patients with rheumatoid arthritis. I found it easier to "reach" the patient when he was in a relaxed state — and that he was more amenable to therapeutic management.

More recently, I have had the opportunity to evaluate the use of hydroxyzine,¶ 10 mg, as an ataractic agent. It has been very gratifying in many cases to note the improvement in sleeping habits, decreased tension and the improved attitude of patients toward their disease and their problems.

This was evidenced by increased initiative and acceptance of responsibility for their own improvement. In an additional study,[5] hydroxyzine, 10 mg, was combined with prednisolone, 5 mg,§ in a group of patients with rheumatoid arthritis who had been maintained on steroids alone for one to three years.

A substantial improvement was noted both objectively and subjectively among most of the patients, as well as increased initiative and responsibility observed with hydroxyzine alone. In some, it was possible to reduce the dosage of steroid without sacrificing the comfort of the patients.

Ataractic Agents

In my experience, these ataractic agents are of great aid in supplementing other therapy in rheumatoid arthritis. However, caution must be exercised so as not to employ the use of steroids with sedation or with ataractic agents in patients when there is even a questionable psychosis present.

The following rules should guide the physician in his approach to the arthritic patient.

The patient should be reassued sufficiently to allay fear, and every effort made to realize a relaxed state in the shortest possible time. Thus, the tense patient will experience some degree of ease so essential to his welfare.

Human Being

The patient is a human being and must be treated as well as his disease. It is the duty of the physician to guide the patient and it is nature's duty to heal.

Individualized treatment is the rule, not the exception. All phases of treatment applicable to the patient should be surveyed, considered and included.

The physician cannot "help" the disease without the patient's cooperation.

Medicine is still basically an art. The physician possesses that art, which is the ability to use and apply this knowledge.

‡Dimethylane (National Drug)—Avacalm®
¶Atarax® (J. B. Roerig)
§Ataraxoid® (Chas. Pfizer & Co.)

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Mucoid Adenocarcinoma: Case Report

By W. R. Gaddis, M.D., *El Paso*

The following case is presented because of its unusual character. Williams[1] has reported 53 cases from his own experience and from a review of the literature.

Shallow, Wagner and Colcher[2] reported 750 cases with age extremes of 16 to 92 years. Cattell, et al[3] reported a series of 164 cases with an age incidence of 31 to 84 years.

It is obvious that malignant disease of the colon is uncommon in the age group under 20 years. These are marked by a high incidence of colloid carcinoma and a high grade of malignancy.

White Male

A white male, born Febraury 13, 1937 was admitted to Hotel Dieu, Sister's Hospital, El Paso, Texas on September 6, 1954 on the service of Dr. Joe Carter with a complaint of pain and swelling of the abdomen for about one month.

He stated that it began with a small lump under his left rib margin about 4 months previously and had been accompanied by intermittent pain and bouts of fever.

He stated that he had lost about 18 pounds in the last five months. There had been a progressive increase in the size of the abdomen with an attendant discomfort. He denied recurrent past history of diarrhea or gastro-intestinal symptoms.

No Bleeding

There had been no bleeding or hemorrhagic diathesis. He had been seen by another doctor locally who had thought he had splenic enlargement due to mononucleosis, or that it might have been a renal mass. This doctor was unable to prove serologic evidence of mononucleosis, and a urological consultation had been unrevealing as to the cause of the swelling and fever.

Past medical history showed the usual diseases of childhood. In the pre-school age he had had a tonsillectomy and an adenoidectomy and an umbilical herniorrhaphy.

Physical Examination

Physical examination on the day of admission to the hospital disclosed a white male of 17 years. He was 5 feet 10 inches tall, weighed 120 pounds and was considered to be in fair nutritional status. His temperature was 99° F, pulse 105 per minute, and blood pressure 120/90, respiration 20 per minute. There was a questional slight icterus of the sclerae. The posterior cervical nodes were palpable. There was dullness to percussion of the lower third of the right posterior chest as if from liver dullness.

The heart was not considered remarkable. The abdomen was diffusely distended and tight, and a fluid wave was elicited. There was a healed transverse mid-abdominal scar replacing the umbilicus.

The spleen was palpable 2½ fingerbreadths below the left costal margin, and there was dullness of four to five inches over the splenic area in the right lateral position.

The other systems were considered to have no significant abnormalities except that "the postcervical and inguinal nodes are palpable and shotty, about as expected normally."

Working Diagnosis

The working diagnosis was malignant lymphoma or aleukemic leukemia.

Admission laboratory work gave the following results:

Urine: Sp.Gr. 1.016, alkaline reaction, albumen and sugar—negative. No red cells present. Blood: Red blood cells 4.34 million with 11.5 gms. hemoglobin. White cell count was 7,300 with 66 segmented forms, two stabs, 22 lymphocytes, six mononuclears, one basophile and three eosinophiles. On the same day an alkaline phosphatase was reported as 4.6 Bodansky units. The serum phosphorous was 3.2 mg/100 cc. The total protein was 7.7 gms. per 100 cc.,5.0 grams of which were albumen and 2.7 grams were globulin fraction with a ratio of 1.8:1. The Van den Berg reaction was reported as direct negative, and indirect 0.30.

X-ray examination of the chest and abdomen at the time of admission showed an accentuation of the pulmonary markings in the right lower lobe, and the abdominal film showed "what appears to be some enlargement of the liver and spleen." No other abnormalities could be demonstrated.

No Bile In Urine

On the second hospital day the reticulocyte count was reported as 0.4 per cent. The urine contained no bile. On the third hospital day the blood

Kahn and Eagle were said to be negative. He was of blood group A.

On the third hospital day the cephalin flocculation test was negative. This same day a paracentesis abdominis was performed with removal of three liters of fluid, 100 ml of which was sent to the laboratory. Report of examination of this fluid was as follows: Specific gravity 1.013, Protein 4.0 mg per 100 cc. The cellular content was 180 white blood cells per mm^3, 320 red blood cells per mm^3, and 290 large cells per mm^3, which the pathologist reported as being of a questionable neoplastic nature.

Re-Examination

A re-check roentgenogram of the abdomen after removal of the fluid was reported as "a very marked enlargement of the spleen. The exact size of the liver cannot be determined due to the presence of the large amount of gas in the ascending colon and distal portion of the stomach."

Re-examination of the urine and blood morphology after four days in the hospital was without significant difference from his admission studies. At the same time it was noted that his ascites was recurring at a rapid rate.

At the end of the first hospital week the examiner stated, "the spleen or tumor mass is definitely fixed and does not move with respiration. A review of the films brings up the question of enlarged spleen, sarcoma, dermoid, teratoma or neuroblastoma."

The following day the pathologist recommended exploratory laparatomy because of inability to identify the tumor cells in the ascitic fluid.

Taken To Surgery

On the 13th hospital day the patient was taken to surgery. Gross findings showed a marked distention of the abdomen. In the upper left quadrant there was firm fixed mass which extended to the mid-line medally and to the level of the umbilicus inferiorly.

There was marked hardening and induration of the transverse mesocolon to the left of the mid-line and extending laterally and superiorly, and inferior from this point there was a large irregular mass.

Laterally it was attached to the anterior abdominal wall and the parietal peritoneum. Scattered over the surface of the peritoneum elsewhere there were numerous implants.

The omentum itself was studded with small nodular areas contiguous with the tumor. A biopsy was taken and the abdomen closed in the usual manner. Postoperative clinical diagnosis was inoperable lymphosarcoma. The pathological report was metastatic mucinous adenocarcinoma of the omentum.

Intestinal Obstruction

The patient's condition continued to detriorate and on the 15th postoperative day intestinal obstruction was diagnosed by clinical and x-ray evidence.

On the 16th post-operative day he was returned to surgery for correction of the obstruction. Transverse colostomy was not technically possible, so a Witzel-type enterostomy was performed in the terminal ileum. His downhill course continued and he expired quietly on the 10th day after the second operation, about 5 weeks after admission.

Post-mortem examination was done on the day of death. The gross anatomic diagnoses were: 1) mucoid carcinoma involving the transverse colon with metastasis to contiguous structures, 2) medastatic-neoplastic implants of the peritoneum, 3) Ascites, 4) obstruction, intestinal, multiple blind loops, 5) pulmonary edema, subacute, 6) gastro-colic fistula, 7, gastritis, subacute, and 8) emaciation.

Microscopic Examination

Miscroscopic examination of the area of the tumor itself showed "extensive neoplasia of the colon characterized by clusters and groups of fairly well differentiated mucinous type cells often lying in an acellular matrix. Mococarmine stains were positive."

The final diagnosis was mucinous carcinoma of the transverse colon with extension to adjacent structures causing final obstruction and death. There was no distant metastasis.

Summary

We have presented a case of mucinous carcinoma of the colon in a 17 year white male. This was a malignancy of the gastro-intestinal tract in which symptoms referable to the intestinal tract were not present, and were mistaken for primary disease of the spleen and liver.

Mills Building

Bibliography

(1) Carcinomia of Colon in Childhood, Williams
Ann. Surg. 139: 816-825, June 1954
(2) Clinical Evaluation of 750 Patients with Colon Cancer, Shallow, Wagner and Colcher
Ann. Surg. 142, August 1955, #2, 164
(3) Cancer of Colon and Rectum, Cattell et al; Surgical Clinics of North America
June 1955, Vol. 33, #3, Saunders.

CURRENT THERAPY

Acute Coronary Occlusion

(continued)

Anticoagulant Therapy

By Jack A. Bernard, M.D., *El Paso*

With a Discussion by Dr. Lester C. Feener, El Paso

After the physician has controlled the initial episode of pain of a coronary occlusion and the complications, he is confronted with the problem of anitcoagulants. Although there are a few who question the value of anticoagulants in coronary occlusion, statistical evidence favors anticoagulant therapy.

In almost 1,000 cases the mortality was 16 per cent in the anticoagulant treated group as compared to 29 per cent in the control group. Thrombo-embolic complications occurred in 41 per cent of the control as compared to 13 per cent in the treated group.

Some Not Benefited

On the other hand there are a few so called "good risk" patients who are not benefited by anticoagulant therapy. Yet in the vast majority of cases these drugs do reduce morbidity and mortality.

However as the period of bed rest is shortened and early ambulation is instituted, the indication for anticoagulant therapy may be diminished, and as time goes on their almost routine usage may not be indicated.

Since there is now apparent unanimity of opinion that anticoagulants do reduce morbidity and mortality and that they are indicated except perhaps in the rare "good risk" patients, the problem is therefore not whether one should use anticoagulants or not, but which anticoagulant should be used.

Therapy Recommended

Routine anticoagulant therapy — the use of anticoagulants except perhaps for the so-called "good risk" patients — is recommended unless there is a definite contraindication, such as the history of a bleeding tendency or some gastrointestinal lesion that might start to bleed. Even in the latter instance anticoagulant therapy may be used with careful control and observation of the patient.

For anticoagulant control, Heparin and one of the oral coumarin drugs is usually used. A simple procedure is to give Heparin 50 to 75 mg. intravenously or intramuscularly every four to six hours for the first 24 to 72 hours or longer, until the oral anticoagulant has produced its effect.

At the same time that the initial dose of Heparin is given, one of the oral anticoagulants is given: for example, Dicumarol 300 mg. the first day then 200 or 100 mg. the second day and thereafter 50 to 100 mg. as a daily maintenance dose.

(I prefer to give the Dicumarol at the same time each day, for example at 10 a.m. In the hospital this facilitates better supervision and easier control. Also it reduces the possible effect of food on absorption.)

Safer Procedure

In the older age group or in a patient in whom one suspects that there might be a bleeding tendency, it may be a safer procedure to use 200 mg. Dicumarol the first day and then 100 mg. the second day and then adjust the dose thereafter according to the prothrombin level, meanwhile continuing the Heparin for the first few days until the prothrombin time is at the desired level.

There are other ways to carry out such therapy depending upon the choice of anticoagulant. These include Coumadin®, Tromexan®, Hedulin®, (or Danilone®) and Cumopyran®. Cumopyran is similar to Dicumarol in its action. Tromexan has a shorter induction period, but control is difficult.

Hedulin (Danilone) has some side reactions. Coumadin is highly recommended, with its prompt

induction period. It has a high predictability of response and is very sensitive to vitamin K or K_1. It may be given intravenously, but it can also be used orally with Herapin parenterally. Usual dosage is 75 mg. orally the first day, then 6.25 - 12.5 mg. daily for maintenance.

Prothrombin Level

As to the prothrombin level, many physicians prefer to use the percentage of prothrombin level in order to control the patient. Others feel that it is much easier and simpler to use the number of seconds.

For example: with the control prothrombin level usually 12 to 16 seconds, many physicians prefer to obtain a therapeutic level in the patient of around 30 seconds. Thus the prothrombin reports should always be in both seconds and per cent and the control should **always** be noted.

Considerable Discussion

There is considerable discussion as to the dangers of anticoagulant therapy. Hemorrhage has been reported as high as 10 per cent with as many as one or two deaths per 100 cases. This seems rather high, but it should be pointed out that in such reports hemorrhage occurred also in five per cent of the control group.

It is true that there is the rare patient who is highly sensitive of anticoagulants. However, in most instances there is little difficulty. If bleeding does occur, vitamin K_1—Mephyton® (Merck) — 50 - 100 mg. intravenously reverses the bleeding usually in three to six hours.

For Heparin overdosage, two per cent Protamine sulfate intravenously milligram for milligram, with or without whole blood is indicated.

Prothrombin Test

As to the prothrombin test, I believe that its difficulty has been overemphasized long enough. With today's commercially prepared thromboplastins, the test is simpler and easier than a blood sugar.

I see no reason why the difficulty of this test should be continuously belabored. It is perhaps better to have the tests run by the same technician and also to run them in groups, **always,** of course, with a control. With a sincere technician, the difficulties of this test are eliminated.

Logical Usage

At first glance the most logical usage of anticoagulant therapy would be for the premonitory stage of coronary occlusion. However, the value of anticoagulant drugs in preventing an impending coronary occlusion has not been borne out by clinical studies.

In reports, patients have been put on anticoagulants in ample time; and in spite of such therapy, with patients under good control, the process has not been stopped and the occlusion was not prevented. At present there is no known way to prevent such attacks.

In many instances it is felt that the occlusion occurs with the onset of pain, with the electrocardiographic changes and other confirmatory findings occurring later.

Actually a little thought as to the value of anticoagulants reveals that their value has not been demonstrated in preventing coronary occlusions. Thus anticoagulant therapy should be continued for at least three to six weeks or longer and then should be gradually tapered off.

Complications Reduced

As regards long term anticoagulant therapy, it has been shown to reduce subsequent myocardial infarctions and thromboembolic complications. In same instances patients have shown an increase in exercise tolerance. It is particularly indicated for the patients that have had more than one infarction and those who have thromboembolic tendencies. Continuous long term therapy is not difficult and after a patient is once regulated on an oral anticoagulant, prothrombin times may be done as little as once a month. The patient is usually maintained at about 23 to 30 seconds in such cases.

Manchester reports the value of continuous (one to 10 years) long term anticoagulant therapy. In his study of 204 patients in the anticoagulant group with 200 in the control group the characteristics of the groups studied were similar. 14.2 per cent of the anticoagulant group had subsequent myocardial infarctions as compared to 34 per cent in the control group. Also the mortality was 20.6 per cent as compared to 53 per cent in the control group. Thromboembolic complications occurred in 5.8 per cent in the anticoagulant treated group as compared to 17.5 per cent in the control group; whereas the mortality in the former was 14.2 per cent as compared to 45.7 per cent.

Interestingly, 51.3 per cent of the anticoagulant group showed improvement to exercise tolerance in walking and in performing the Master's two-step as compared to 27.5 per cent improvment in the control group.

The mortality was 5.4 per cent in the anticoagulant series as compared to 41.6 per cent in the control series.

Hemorrhagic complications occurred only in the anticoagulant group and this was 2.9 per cent with no fatality.

Summary

Anticoagulant therapy is recommended for most patients with coronary occlusion. The occasional

"good risk" patient may be spared but it is felt that even in such cases anticoagulant therapy should be started and then tapered off as the patient becomes ambulatory.

The choice of anticoagulant is an individual one. Coumadin is highly recommended.

The test for prothrombin activity is not difficult and its difficulty has been overemphasized.

Anticoagulant therapy may be tried for the premonitory stage of coronary occlusion. Its usage and indication seems logical but so far such efforts have not prevented the occurrence of occlusions in such cases.

Long term anticoagulant therapy is not difficult and is recommended in patients who have had multiple attacks and particularly those who have had phlebothrombosis or embolic phenomena. Angina is reduced in some instances.

As the period of bed rest is shortened and early ambulation is encouraged, the need and indication for anticoagulants may diminish.

Discussion by Dr. Feener

The essayist has presented the views which are held throughout the United States today. Anticoagulant therapy is given to a majority of people who have suffered acute myocardial infarction. The contraindications to this therapy have been outlined adequately.

It must, however, be remembered that there are a distinct minority who are not as enthusiastic over anti-coagulant therapy as the majority. It is difficult to evaluate the efficacy of this particular type of therapy, and opinions will naturally vary.

Caution Urged

One should be cautious in talking and dealing with the lay public on this subject. There are many men in the rural areas who have treated myocardial infarction for many years successfully without anti-coagulant therapy, and will continue to do so.

These men are competent and have an absolute right to their opinion, and their opinion should be respected. Furthermore, if the internist or cardiologist treats coronary thrombosis with resulting myocardial infarction for a period time, he will at some time become very suspicious that his anticoagulant therapy has become detrimental rather than helpful to the patient.

Two-Edged Sword

I do not know of many cardiologists or internists who are honest with themselves who have not at least thought possibly they have seen this phenomenon. It behooves each one of us to remember that anti-coagulant therapy is a two-edged sword, and that it must not be considered lightly.

Last but not least, be cautious in your criticism of the practitioner who does not agree with you on the use of anti-coagulants, because there are times when anti-coagulant therapy does not arrest thrombo-ambolic phenomena whether associated with myocardial infarction or not. It must be remembered that the last word has not been said on anti-coagulant therapy, even with the addition of the new hypoprothrombinemic drugs.

Postgraduate Session Scheduled in El Paso

Dr. George E. Schreiner, professor of medicine at Georgetown University, Washington, D. C., will be the visiting speaker at a session of El Paso Branch of the Postgraduate School of Medicine of the University of Texas beginning at 8:30 p.m. Sunday, February 23, and running through the day in the Turner Home of El Paso County Medical Society, 1301 Montana Street.

Members of the American Academy of General Practice will receive seven hours credit for attending the session, Dr. Ralph H. Homan, dean of the El Paso Branch of the Postgraduate School, said.

Physicians from New Mexico, Arizona and Mexico as well as from Texas are invited to attend, Dr. Homan pointed out.

Dr. Schreiner will speak on "Edema States" at 10:40 a.m. and on "A New Diuretic — Diuril," at 2:30 p.m.

All other speakers will be from El Paso. Their subjects and times follow: Dr. M. Nathan Kleban, "Diabetes and Acidosis," 8:30 a.m.; Dr. William I. Coldwell, "Cardiac Decompensation, 9:10 a.m.; Dr. John M. Verosky, "Diarrhea and Dehydration in Infants," 10:40 a.m.; Dr. H. W. Demarest, "Eclampsia and Toxemia of Pregnancy," 1 p.m.; Dr. Charles E. Webb, "Surgery," 1:40 p.m.; and Dr. W. P. Stratemeyer, "Care of Head Injuries," 3:10 p.m.

Edited By ANDREW M. BABEY, M.D., LAS CRUCES, N. M.

APHORISMS and MEMORABILIA

Truths and Concepts Concerning the Bronchopulmonary System

1. "Localized wheezes are never due to asthma."—R. KERN, *International Clinics*, 3: 183, 1936.

2. "I—do not believe in disturbing this (traumatic) hemothorax unless is is absolutely necessary."—RALPH BETTMAN, *loc. cit.*, page 667.

3. "Morphine should be used in asthma only as a last resort. Its effect is that of a general depressant. It does not relieve bronchospasm, except in cardiac asthma."—F. RACKEMANN, *Clinical Allergy*, The MacMillan Company, New York, 1931, page 468.

4. "There is only one dust that may be dangerous and that is the dust which contains silicon in some form. The organic dusts and those inorganic dusts which do not contain silicon are harmless."—JOHN HAWES, *New England J. Med.*, 216: 163, 1937.

Lung Abscesses

5. "Most lung abscesses that we see die of one of three things:—1—a fatal hemoptysis, 2—extension of the infectious process to the other lung with rapidly spreading septic pneumonia, 3—penetration into the pleural cavity with empyema."—TRACY MALLORY, *New England J. Med.*, 205: 690, 1931.

6. "Egophony is one of the most unreliable and useless signs."—RICHARD CABOT, *New England J. Med.*, 199: 1219, 1928.

7. "Miliary tubercles do not give physical signs, but they often do produce cyanosis."—RICHARD CABOT, *New England J. Med.*, 198: 408, 1928.

8. "The presence of active tuberculosis and asthma in the same patient is a rarity."—GEORGE BRAY, *Recent Advances in Allergy*, 3rd. Ed., P. Blakiston's Son & Co., Philadelphia, 1937, page 129.

9. (Cell count in chest fluids)—The count varies. "If you tap the upper portion you may get no cells, if the lower portion, many cells."—TRACY MALLORY, *Boston Med. & Surg. J.*, 195: 1032, 1926.

10. "The pain of pleurisy is capable of very extensive reference—it may be felt as high in the body as the cervical vertebrae and as low as Poupart's ligament."—LORD HORDER, *Medical Notes*, Oxford University Press, London, 1921, page 52.

Differential Diagnosis

11. "It should be emphasized again that the blood forming a hemothorax may come from the vessels of the chest wall as well as from any of the thoracic contents. It is important to make this differential diagnosis, if possible, from the standpoint of therapy. Hemorrhage from the thoracic contents rarely calls for operative treatment, whereas bleeding intercostal and internal mammary vessels must be caught at once."—FRANK BOLAND, *Proceedings of Interstate Post Grad. Med. Assembly of North America*, Freeport Press, Freeport, Illinois, 1931, page 356.

12. "The asthmatic should go supperless to bed."—J. A. LINDSAY, *Med. Axioms, Aphorisms & Clin. Memor.*, H. K. Lewis & Co., London, 1923, page 5.

13. "Pneumonia in the aged may give the appearance of cerebral apoplexy—even with the aspect of true hemiplegia."—CHARCOT, *Clin. Lectures on Senile & Chronic Diseases*, New Syndenham Co., London, 1881, page 38.

14. "Calcification is usually looked upon as a measure of healing of tuberculous lesions. It should be remembered, however, that only the caseous parts become infiltrated with calcium salts; in other words, this is a reaction in necrotic tissue which harbors, or has harbored numerous bacilli, rather than an indication primarily of healing capacity. Therefore, I look on the extent of calcification as an index of the extent of pre-existing caseation, realizing that such foci, although appearing hard on the X-ray films, may still be rather cheesy or chalky and capable of liberating tubercle bacilli."—J. B. AMBERSON, JR., *New England J. Med.*, 219: 575, 1938.

By W. Compere Basom, M. D., El Paso, Orthopaedic Editor of Southwestern Medicine

ORTHOPAEDIC SURGERY NOTES

It is beginning to be felt that most scolioses with absence of vertebral deformity are actually due to sub-clinical cases of poliomyelitis. Therefore, after an upper respiratory infection with backache, patient should have the spine x-rayed for several months thereafter.

Children with foot deformities should have their pelvis x-rayed inasmuch as the hip joints may be deformed. This may not be obvious on clinical examination.

With experience and time the hip prosthesis situation is improving. On carefully selected cases the percentage of success is nearer 80 per cent than 50 per cent previously reported.

Newer Techniques

Arthrodesis of the hip joint on the other hand is a good procedure and newer techniques and technical advances are being frequently reported.

In slipped capital femoral epiphyses early diagnosis is very important in aiding the obtainment of a good result. The earlier the case can be "picked up" the better the result.

In fractures of the femoral neck and intertrochanteric region in children, the more conservative treatment is consistent with best results. In other words, closed reduction, blind pinning, casts and traction are in order. In contrast, older patients do better with the open reduction or closed reduction and nailing.

For application of long leg casts on the lower extremity in using a fracture table the pressure of the suspension back of the knee and about the ankle should be distributed over a wide area.

Large Felt Pad

In other words a large felt pad placed about the knee and the ankle, then application of muslin suspension is best. Sheet wadding can then be applied over the entire area and the cast applied leaving the front or anterior part of the knee open so that after the plaster sets, the muslin bandage can be completely slipped out.

This will eliminate any possibility of pressure in that region.The foot had best be treated separately, removing the foot traction and substituting manual traction. Remove the muslin bandage.

Then the application of the plaster while the foot is held manually and the knee is still suspended will eliminate the possibility of pressure sores about the ankle.

Several layers of sheet wadding under the cast certainly facilitates the removal of the cast with the minimum danger of cutting or pinching the patient's underlying skin.

Eighty per cent of people with spondylolisthesis have a familial history. Fifty-two per cent of Eskimos have spondylolisthesis. The incidence of spondylolisthesis rises steadily from infancy to complete bone maturity. Embryologically, the bases of the two facets and the isthmus of the lumbar vertebrae all originate from one area and not separate growth centers.

A dark spot in an area of Paget's disease should lead one to suspect malignancy when roentgenograms are reviewed. Biopsy is then in order if possible. Mortality rate of malignancy in Paget's disease is 95 per cent.

In fractures of the articular surface of the os calcis, if there is displacement present, open reduction will show usually far more displacement than was discernible by x-ray. After reduction by open procedure, pull-out pins can be used successfully to hold the fragments in position until union occurs.

Tibialis Posterior

The tibialis posterior is receiving more and more attention in the management of recurrent club foot.

In the management of scoliosis, early fusion and re-fusion in case pseudarthroses develop, is advocated.

Growth stimulation operations for leg inequality are becoming more popular and are felt at this time to give an increased amount of growth with each procedure.

In this day and age one sees particularly in some of the manufacturing fields apparently anything for a change. In other words, another model is put out merely for the sake of having a new model and not all of the so-called advances are definitely advantages over what was already being produced.

We must not let this influence us in the practice of medicine. We should continue to use procedures which have been tried and found to be best until the so-called new procedure definitely has demonstrated and definitely has been proved over a period of adequate time to be definitely superior. There, of course, are some situations in which the new procedure may very obviously outclass the standard or older one and definitely deserve use.

Next Month: A Crash Report on the latest research.

THE PRESIDENT'S COLUMN

Suggestions on Purposes and Goals of Southwestern Association Urged

By Louis G. Jekel, M.D., *Phoenix*

Last month this column carried a general outline of the purposes and goals of the Southwestern Medical Association. Over a period of years these functions have met the needs and desires of our members.

We want this happy state of affairs to continue. If the members in accordance with the times prefer changes in the procedures and programs it should be possible for them to express their views and effect all such desired changes. Fortunately, it is possible for them to do so.

The Association is operated in a democratic manner. At each annual session a business meeting is held. This meeting parallels a stockholders meeting. An executive committee (Board of Directors) and a slate of officers are elected to manage the Association for the ensuing year.

Controlled By Clique

If ever it is thought that the Association is controlled by a clique, it may be true. The reason is that only a quite small number of members take an active interest in the proceedings. All members are urged to participate but they do not do so. Nevertheless it is possible for the members to express opinions, voice protests, and generally control the entire set-up if they so desire.

In addition to actual control of the organization members have another means at their disposal to get what they want. They may request special subjects for discussion at the annual meeting. The program committee is always anxious to produce a beneficial program that will be popular with the audience.

Requests Will Be Filled

Requests for subjects will be fulfilled whenever possible. It must, of course, be remembered that the program is prepared about a year in advance, so suggestions and requests should be submitted with that fact in mind.

It is my belief that the Southwestern Medical Association would benefit greatly from active participation by its members. Such activity would lead to renewed vitality in this venerable Association, and would redound to the benefit of each member.

Dr. C. R. Swackhammer

Physicians who attended the recent annual meeting of the Southwestern Medical Association in El Paso expressed deep regret at the death during 1957 of Dr. C. R. Swackhammer, 71, of Globe, Ariz.

Dr. Swackhammer, until stricken by a prolonged illness of five years, was a regular attendant at Southwestern Medical Association meetings and was vice-president of the Association for three years, 1935-37.

MONTHLY CLINICAL PATHOLOGICAL CONFERENCE
EL PASO GENERAL HOSPITAL

November 21, 1957

FREDERICK P. BORNSTEIN, M.D., EDITOR — Case Number 915

Presentation of case by Ward Evans, M.D.

History—Dr. Nathan Kleban:

Abdominal pain and shock brought a 48-year-old Latin-American mother of seven surviving of 16 children to the hospital on September 8, 1957. She died on September 11, 1957.

Twenty-four hours before admission, at 1:00 P.M., the patient experienced severe mid-upper abdominal pain which spread to the back. Three hours later, she began to vomit small amounts of yellow liquid material. Because of persistence of pain, recurrence of vomiting, and development of shock, the patient was sent to the hospital.

Except for the notation that the patient's general health and appetite had been poor, there was no oher significant contributory history recorded.

Physical Examination

T. 104.6 (R.) R. 32. On admission to the emergency room, there was no detectable peripheral pulse. Skin was cold. Heart sounds were distant. There was generous abdominal fat, slight rigidity, no bowel sounds, slight upper abdominal tenderness.

Hospital Course

Plasma and 1EVO-arterenol were started through a venous cut-down. Continuous gastric suction was begun. An indwelling catheter was inserted into the urinary bladder.

Except for a brief period, it was necessary to maintain the blood pressure with 1EVO-arterenol. Rectal temperatures ranged from 102 to 105. The patient was generally restless. She pulled out her gastric tube and I-V catheter or needle repeatedly.

Medications were Probanthine, Penicillin, Streptomycin, Tetracycline, Sparine, Aspirin, ACTH, Meperidine, Papaverine, Cedilanid, Phenobarbitol. Calcium gluconate and potassium chloride were added to I-V solutions. Arterenol-containing solutions infiltrated from cut-downs.

Urine out-put was 500 cc. on the first day, 2260 on the second, and 1600 on the third. On the third day there was a chill, dry cough, and cyanosis. Shortly before death rectal temperature rose to 107.

Respirations ceased before the heart stopped beating.

Laboratory Findings

Blood counts: September 8, 1957—Hb. 15.6 gms., Ht. 53%, WBC 13,900, Stab. 19, Segs. 63, Lymph. 18. September 9—Hb. 12.9 gms., Ht. 50%. Sept. 8—Hb. 12.4 gms., Ht. 48%. September 9—Hb. 12.0 gms. Ht. 45%, WBC 8,500, Stab. 2, Segs 78, Lymph. 20. September 10—Hb. 10 gms., Ht. 38%.

Serum amylase: September 8—560 units. September 10—285 units.

Urinalyses: September 8—S. G. 1,024-acid, WBC 40-50, occ. WBC clumps, albumin and sugar negative, 4-6 Squamous Ep. cells, bacteria, field packed. September 9—Bile-negative. September 11—S. G. 1.017, Albumin 2+, Sugar 1+, acid, few WBC, many RBC, granular casts, packed, amorphous urate crystals, much mucous, few vesicle Ep. cells.

Blood chemistry: Chlorides, September 10—87 mEq./1. CO_2—19.3 mEq./1. Vanden Bergh Direct—.6 mg.%, Indirect—1.4 mg.%, NPN—24 mg.%, Potassium — 4.2 mEq./1, Sodium 140 mEq./1.

Cephalin Flocculation: September 11 — 24 hours, 3+; 48 hours 4+.

Blood culture: Negative (at four weeks)

Serology: September 8—Negative.

X-rays: September 9—Upright abdomen, no free air observed under either hemi-diaphragm—findings consistent with ascites. September 9—Cholangiogram following intravenous administration of cholografin shows non-visualization. September 11—Chest (portable) unsatisfactory.

Electrocardiogram: September 8—Sinus tachycardia with rate of 150. Extensive myocardial change is probably secondary to shock.

Differential Diagnosis: Dr. Ward Evans

The history states that this 48-year-old white woman had a sudden onset of upper abdominal pain, radiating to the back, 24 hours before admission. It was accompanied by circulatory collapse.

Physical examination revealed a cold and clammy skin, and there were distant heart sounds. Examination of the abdomen revealed it to be rigid and tender in the upper half and silent to auscultation. The patient was obese.

Having proceeded this far with the examination, it is possible to draw a few conclusions.

First of all, the patient had a peritonitis of rather sudden onset, and there would be three main reasons to account for this:

1. A perforated ulcer,
2. an acute gall bladder attack, or
3. an acute pancreatitis.

Other conditions less likely would be a coronary thrombosis, a pneumonia simulating peritonitis, or, possibly, a high lying appendix. However, these latter diagnoses are not very probable with such a sudden onset as occurred here.

Impression Confirmed

Laboratory procedures confirmed the impression of peritonitis with a white count of 14,000 and 80% granulocytes. An x-ray of the abdomen did not reveal free air under the diaphragm. The urine was essentially normal except for a few white cells which are of no significance.

The serum amylase was elevated to 560.

The elevated serum amylase is the most significant finding in this case and permits you to crystallise your opinion: namely, that the patient most likely had an acute pancreatitis.

There is no ulcer. The history makes an acute gall bladder attack unlikely and an acute gall bladder attack would not account for an elevated serum amylase.

Therefore, we now have to differentiate between the various factors that might cause an elevated serum amylase.

Certainly by far the most common cause is an acute pancreatitis.

A posterior penetrating ulcer might also produce it, as would any acute inflammatory process in the upper abdomen which would involve the pancreas, directly or by extension.

A process by extension would most likely be a penetrating ulcer from the stomach or duodenum, but it does not seem likely to me in this case.

So, I think the doctors handling the case were justified if they considered the case an acute pancreatitis and treated the patient accordingly.

Pancreatitis Regimen

The treatment was that of a pancreatitis regimen, plus maintenance of liquid and electrolytes plus splachnic block.

Nevertheless, the patient died three days after admission which, with this particular disease, I consider the common outcome. The diagnosis, then, is acute hemorrhagic pancreatitis.

Dr. Jack C. Postlewaite

The brevity of the case probably makes it more difficult, because there are many things we would like to know in her past history that might have contributed to a diagnosis.

For example, perhaps she had gall bladder disease.

Non-visualization of a gall bladder is certainly not impossible with an acutely ill patient with electrolyte and fluid shifts such as she had with her acute course of three days.

I am impressed that they did do a gall bladder series, inasmuch as many of her liver function tests were abnormal and the intravenous study is a rather difficult study at best.

The fact that they did it, though, at least made the house staff and the attending physicians look in the direction of liver, gall bladder and pancreas.

I don't doubt the diagnosis of pancreatitis, probably acute, edematous to begin with, perhaps of a viral or toxic origin and subsequently hemorrhagic with fatal termination.

One gets the impression that other problems exist.

For example, albuminuria and glycosuria occurred which is not unlikely in a progressive pancreatitis.

Perhaps surgical intervention was indicated, although it adds to the morbidity and mortality of these cases and most surgeons prefer not to go in.

However, she had ascites by x-ray and one wonders why a needle couldn't have been introduced and the ascitic fluid analysed for other elements than fat necrosis and pancreatic secretions.

Then you get the impression that another event occurred, something pulmonary.

Was an embolus possible?

Now the pancreatic lesions, malignant lesions, for example, are associated with the thrombo-embolic phenomena.

Unexplained peripheral thrombosis should always be investigated for the possibility of a pan-

creatic lesion, usually malignant, rather than pancreatitis.

Myocarditis

The only other comment: one is interested in the myocarditis diagnosed apparently by her vasomotor collapse and diagnosed by her electrocardiogram.

Is this electrolyte or is this organic lesion of the myocardium?

Pancreatitis is a disease with a liberation of high amounts of amylase, which produces hemorrhagic phenomena.

It is not limited just to the viscera where there is fat necrosis.

It is circulated in the fluids and often gives hemorrhagic myocarditis and hemorrhagic encephalitis with petechiae, round cell infiltration, perivascular cuffing, and so forth.

These people have myocardial findings as well as encephalitis type terminal symptoms.

Dr. Manley R. Cohen

The only comment on the chest film, which is a portable, is that the film was unsatisfactory. Even though it is unsatisfactory, there are a few changes in here which point to some type of pulmonary pathology.

Regardless of the diagnosis, certainly there is atelectasis at the right base, and some fluid on the left.

With a patient suspected of acute pancreatitis, this is not at all surprising, as pointed out by Dr. Postlewaite.

Certainly from the x-ray point of view, pulmonary emboli are almost a must in considering a secondary diagnosis.

Dr. Frederick P. Bornstein

Dr. Postlewaite mentioned congestive heart failure. Does congestive heart failure develop in three days? Is that possible?

I always thought that congestive heart failure develops on the basis of a more or less longstanding disease.

When a heart gets functionally impaired very quickly, do you really develop congestive heart failure in the proper sense of the term in three days?

Dr. Cohen

Yes.

Dr. Saul B. Appel

That's an interesting question, but congestive heart failure certainly can develop in a matter of hours to a day. Of course, microscopically, you may not see the same picture, with the pigment-laden macrophages in the lungs and the congestive changes in the liver, but the clinical picture of congestive heart failure can develop very quickly.

What I was thinking of, is that while we are all looking at the upper abdomen in this patient, actually, the picture is non-specific, and I think it is possible to conceive of almost anything rupturing within this abdomen and, in the supine position, the fluid could travel upwards under the diaphragm and also produce severe pain in the back which might not have been localized.

So acute pancreatitis seems a very likely possibility, but as long as this is a CPC and nobody else has mentioned it, I think we should mention rupture of an ovarian cyst, tumor, or possibly, even rupture of an abdominal aneurysm. However, I doubt very much that we would have this picture, but still, I think that anything that ruptured within the peritoneum, or even outside the peritoneum, could produce a picture somewhat like this.

Amylase Figures

The two amylase figures do not impress me very greatly, even though they were done at this hospital.

I think Dr. Dow and I remember a patient with an amylase of 3200, just a few weeks ago at another hospital, and then the next day it was down to 80, and he did not have pancreatitis.

But it was 3200, and the technicians swore that this was, very obviously, a high amylase, so I wouldn't stick my neck out and say this means pancreatitis. As Dr. Postlewaite says, there are other things.

Dr. Evans mentioned a penetrating ulcer, or perhaps anything that is sitting above the pancreas could produce, by irritation, a serum amylase this high.

Dr. Pablo Ayub

I think there is probably no doubt that this patient had peritonitis, and whether the pancreatitis could be the primary lesion or not is questionable.

This could begin, as Dr. Appel said, some other place in the abdomen, even with an appendix, or any other type of bacterial infection.

If the appendix were to burst, an abscess with secondary thrombophlebitis, a pylephlebitis could then give you a secondary pancreatitis from just plain thrombophlebitis.

This portal phlebitis could conceivably cause clots which could detach, and some small clots could find themselves in the lungs.

Dr. Bornstein

If you once have a septic abscess in an organ,

you can develop loose thrombi in adjacent vessels which produce emboli.

If your portal system gets thrombotic, you can develop abscesses in the liver.

You cannot have direct pulmonary emboli from a thrombus in the portal vein.

There has to be an additional septic focus inside the liver to produce pulmonary emboli.

Dr. E. S. Crossett

One other guess hasn't been specifically made.

Dr. Ayub pointed out that an abscess in any part of the abdomen might cause a picture like this.

There is a little note there that the woman had been ill, and had a poor appetite for some time.

There is evidence of liver damage, and the fluid in her right base could be on the basis of infection under the diaphragm — a liver abscess or subdiaphragmatic abscess.

Clinical Diagnosis: Acute hemorrhagic pancreatitis.

Dr. Ward Evans Diagnosis: Acute hemorrhagic pancreatitis.

Pathological Diagnosis: 1. Acute hemorrhagic pancreatitis. **2.** Abscess of minor omental bursa with perforation and retroperitoneal abscess. **3.** Pleural empyema on the left.

Pathological Discussion—Dr. F. P. Bornsteni:

Well, the autopsy findings more or less cover the entire body in this particular case. The peritoneum was dull and hyperemic and there were all the signs of an acute perotinitis. The basic lesion was that of an acute hemorrhagic pancreatitis.

Everybody here made this diagnosis. Due to the necrosis of this pancreas, a large abscess had formed in the minor omental bursa.

Abscess Perforated

This abscess had perforated into the retroperitoneal space and had produced a large abscess on the left side between the spleen and the diaphragm.

Directly above it, on the left side, was a large empyema of the left pleural cavity with collapse of the left lung.

So we have lesions extending into all directions, into the peritoneum, into the retro-peritoneal space, into the thorax.

It all started out with an acute hemorrhagic pancreatitis.

Gall Stones

The patient did have gall stones.

I would have been delighted to find a myocarditis, but did not.

I think in this particular case the general shock, peritonitis and the electrolyte disturbance are sufficient to explain the cardiac symptoms.

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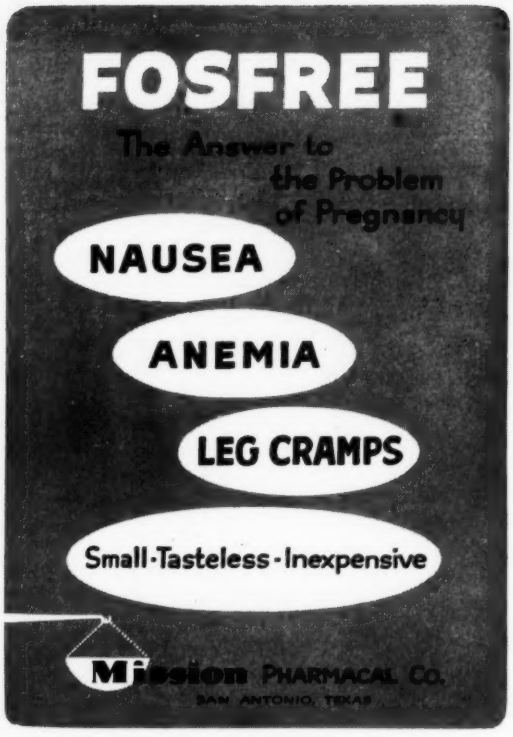
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